Perspectives in Occupational Dermatology

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Because large surface areas of the skin are exposed directly to the environment, skin is an organ particularly vulnerable to occupationally induced disease. Statistics show that, excluding accidental injury, nearly half of all occupational illnesses occur in this organ; a fourth of all workers suffering from occupational skin disease lose an average of 10 to 12 workdays. The constant evolution of new industrial chemicals and methods of manufacture continue to bring new skin hazards and disease into the workplace. Occupational health physicians and practitioners, who usually have minimal training in dermatology, must diagnose and treat unfamiliar diseases in a setting of even less familiar, often overwhelming, technology. A thorough understanding of cutaneous defense mechanisms, clinical patterns of occupational skin disease and methods for establishing accurate diagnoses is essential.

The first record of occupational skin disease may be attributed to Celsus, who described skin ulcerations from corrosive materials in 100 AD. About 1500 AD, Paracelsus and Agricola described cutaneous ulcerations from metallic salts in miners. Little else was recorded until 1700, when Ramazzini wrote his first major treatise on disease in tradesmen. In 1775 Percival Pott published the first description of an occupationally induced neoplasm, cancer of the scrotal skin in chimney sweeps who had been in contact with soot.[1]

The first major treatise on occupational skin disease, *The Dermatergoses,* was published in 1915 by Robert Prosser White,[2] an energetic English general practitioner. The detailed clinical descriptions of occupational skin diseases in this book, particularly the description of irritant dermatitis, continue to be useful today. Publication of *The Dermatergoses* undoubtedly had some role in prompting the United States Public Health Service to study skin disease in industry, culminating in the publication of *Occupational Diseases of the Skin* by Schwartz, Tulipan and Peck in 1939.[3] The last edition of this book was published in 1957.[4] In a field undergoing frequent and dramatic technological change, recent smaller texts provide excellent but limited information on various subject areas.[5-7]

Excluding accidental injuries, which account for approximately 97 percent of occupational disease, skin disease constitutes almost 50 percent of the remaining occupational illnesses.[8] This is not surprising because the skin, with its large surface area in direct contact with the environment, is one of the most vulnerable organ systems. Approximately one of every four workers afflicted with occupational skin disease loses time from work, an average of 10 to 12 days.[8] This represents an enormous loss of productivity each year. The highest relative risk occurs in the manufacturing and agricultural industries, where workers are four times more likely to have occupational skin disease than in other industries.[8]

Cutaneous Defense Mechanisms

The skin is endowed with a host of versatile mechanisms to defend itself against environmental and occupational insults and stresses. These include a barrier to shield against penetration by chemical substances and prevent fluid loss from the body, buffering action, immunologic defense mechanisms, resistance to mechanical friction and trauma, cooling mechanisms to reduce thermal stress, protection against solar radiation and defense against attack by microbial agents.[9]

The Cutaneous Barrier

The cutaneous barrier may be considered from anatomical, biochemical and physiological viewpoints.[10]

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Since the classic water loss experiments of Windsor and Burch almost 40 years ago,[11] it has been recognized that the principal barrier to percutaneous penetration resides in the outermost layer of skin, the stratum corneum. This 15- to 30-μm layer consists of flat, hexagonal cells, containing keratin and other structural proteins. It covers most body areas; its depth varies with the body regions. It is only a few cells thick over eyelid and genital skin, but approaches 300 μm on the palms and soles. The intercellular spaces are filled with a nonpolar lipid material, and may be the principal route of penetration for some substances.[12] Damage or removal of the stratum corneum significantly enhances permeability to a variety of penetrants.[10] The protective function of this anatomic barrier may be further subserved by the eccrine sweat glands, the activity of which may wash off and dilute any noxious substances that come in contact with the skin.

Biochemically, 50 percent to 60 percent of the stratum corneum is structural cytoplasmic protein, 20 percent to 25 percent water and 15 percent to 20 percent lipid. Changes in the water content through entrapment of water on the skin surface or prevention of evaporation (hydration), or environmental chapping (dehydration), may dramatically increase permeability. Alteration in protein or lipid content through detergent or solvent action may produce a similar effect. Qualitative changes in cutaneous lipids may further alter cutaneous permeability. Diets deficient in essential fatty acids produce scaling of the skin associated with pronounced increases in transepidermal water loss.[13]

The physiological basis for percutaneous penetration has been most frequently expressed in the equation known as Fick's law:

$$J = (DK/\lambda)\Delta C$$

in which J equals flux of the penetrating molecule, D represents the diffusibility of the penetrating molecule in the stratum corneum, K represents the partition coefficient (that is, the ratio of the solubility of a substance in the stratum corneum to its solubility in the applied vehicle), λ equals thickness of stratum corneum, and ΔC equals concentration difference of the penetrating molecule across the stratum corneum.[10] While the mathematical laws governing penetration are in reality more complex, the above relationships are useful in understanding and predicting qualitative changes in penetration. Changes within the stratum corneum that enhance diffusibility, vehicles for penetrating molecules that result in increased solubility in the stratum corneum relative to the vehicle, cutaneous exposures on surfaces where the stratum corneum is thinnest and increased concentrations of the penetrant may all enhance absorption over a finite period of exposure. These considerations become important when a physician is evaluating occupational exposures associated with cutaneous or systemic signs and symptoms.

Buffering Action

The skin offers some protection against exposure to alkaline substances by virtue of the buffering action of the outermost layer, sometimes called the "acid mantle." Lactic acid, amphoteric amines and weak bases are deposited on the surface, either through breakdown of the superficial cell layer or by eccrine sweat. Diffusion of carbon dioxide through the skin may further enhance the buffering action.

Immunologic Mechanisms

Immunologic defense mechanisms allow the body to react defensively to chemical stimuli, potential carcinogens and infectious agents. Both immediate (humoral) and delayed (cellular) immunity may be involved. The activities of the immune system, while normally protective, occasionally may have deleterious cutaneous effects.

Immunologic urticaria (hives) results when IgE-coated cutaneous mast cells release histamine and other vasoactive substances, following combination with the appropriate antigen. Occupationally induced urticaria is usually a secondary reaction to inhaling antigen,[14] and is often associated with other symptoms of inhalant allergy. Localized urticaria may result from direct percutaneous penetration by antigen. This phenomenon has been dubbed "contact urticaria."[15]

Delayed hypersensitivity results in a sequence of inflammatory processes collectively known as allergic contact dermatitis. Following percutaneous penetration, the antigen (hapten) must combine with a skin component (protein) before it can be recognized as antigenic. The specific carrier protein is unknown but may differ, depending upon the nature of the allergen and the individual person. Presumably, it is the hapten-protein conjugate that is ultimately recognized as antigenic. Thus, simple metallic substances such as nickel, or even low-molecular-weight lipids and carbohydrates, may occasionally cause allergic contact dermatitis. Present evidence suggests that the initial step in recognition involves selective uptake by epidermal Langerhans' cells before transport to regional lymph nodes.[16] When sensitization occurs, T cells proliferate in the paracortical areas of regional lymph nodes. Upon reexposure to antigen, these sensitized T cells release mediators (lymphokines) in the skin, resulting in inflammation. Suppressor T cells may further modulate this delayed hypersensitivity response.

Mechanical Resistance

The dermis, situated beneath a relatively thin epidermal covering, is composed chiefly of connective tissue cells and fibers as well as a matrix of ground substance. The tough fibrous and elastic components form a resilient barrier against mechanical friction and trauma, and support overlying epidermis, blood vessels, appendages and neurosensory elements. The dermis plays a prominent role in wound repair.

Cooling Mechanisms

Loss of radiant heat from the vascular bed of the skin, by virtue of mechanisms that alter blood flow in the superficial capillary network, plays an important thermoregulatory role when a person is exposed to environmental conditions that produce skin surface temperature changes between 25 and 31° C. Above these skin surface temperatures, further thermoregulation is accomplished by evaporative cooling through activity of the eccrine sweat glands.

Protection Against Ultraviolet Radiation

The protective pigment in the skin (melanin) is synthesized in the melanocytes, located along the basal cell layer of the epidermis. The pigment is "packaged" within the melanocytes into granules called melanosomes. Under suitable stimulation from ultraviolet light, these granules are dispersed into neighboring keratinocytes, resulting in pigment darkening and enhanced protection from ultraviolet radiation. The starting point of melanin synthesis is the amino acid tyrosine, which is converted by the copper-dependent enzyme tyrosinase into dihydroxyphenylalanine (dopa). From here, melanin synthesis proceeds through a number of other intermediate steps, resulting in dopaquinone, 5,6-dihydroxyindole and indole-5,6-quinone before the final melanin polymer is formed. The melanocytes may be specifically or nonspecifically injured as a result of occupational exposures, resulting in pigmentary changes.

Microbial Defense

Intact skin is a highly resistant physical barrier to microorganisms and is infected with great difficulty. Free fatty acids, derived from enzymatic degradation of sebaceous gland lipid, may also have antifungal and antibacterial action. Odd-numbered C5, C7, C9, C11 and C13 fatty acid chains have more potent antifungal activity than even-numbered chains of the same length. Long chain unsaturated fatty acids have limited bacteriostatic activity against a small group of organisms. Once skin penetration by microorganisms has occurred, both immune and nonspecific inflammatory mechanisms provide a second line of defense.

Clinical Aspects of Occupational Skin Disease

The skin is a barrier to the systemic absorption of toxic chemicals as well as a target organ for disease.

Percutaneous Absorption

Skin absorption is an important route of entry for many substances that may produce systemic toxicity. Chemicals that are detoxified by a first pass through the liver following intestinal absorption may be more toxic when absorbed through the skin.[17] Skin absorption may be the principal route of exposure for many agricultural pesticides.[18]

Development of symptoms depends partly on the ability of a substance to penetrate through skin, as well as the inherent toxicity and quantitative degree of exposure. Any circumstance or condition of exposure in the workplace that increases percutaneous absorption may enhance toxicity. Such conditions include increases in stratum corneum hydration, particularly when substances are occluded against the skin by water-impermeable clothing (such as rubber gloves); dry, chapped skin; skin already damaged by cuts, abrasions or preexisting dermatitis; elevation of skin surface temperature, especially through heat transfer from heated industrial liquids; a favorable stratum corneum:vehicle partition coefficient, and contact with anatomical areas of skin that are more permeable (for example, eyelid, facial and genital skin).[19,20] Toxicity also depends on the total amount of exposure, which can be increased through changes in concentration of the penetrating substance and increased duration and frequency of skin contact. The interplay of the above factors on skin absorption of pesticides has been studied by Maibach and Feldmann.[21]

A partial list of substances that may produce toxic symptoms in industry following percutaneous absorption includes aniline dye (methemoglobinemia), benzidine (carcinoma of the bladder), cyanide salts (cellular asphyxia and death), organophosphates (cholinesterase inhibition, gastrointestinal, cardiovascular and neuromuscular disturbances) and other insecticides, and numerous solvents such as methyl butyl ketone (central nervous system depression and peripheral neuritis).[22] Solvents may enhance their own toxicity by damaging the stratum corneum and increasing their percutaneous absorption. Visible inflammation (dermatitis) is not a necessary prerequisite for significant percutaneous absorption. However, substances that also produce dermatitis, such as cyanide salts,[23] may enhance their own systemic toxicity.

Contact Dermatitis

Approximately 75 percent of all occupationally acquired skin disease is "contact dermatitis." This term refers to an inflammatory condition of the skin (eczema) induced by external contact between a substance or material and the skin surface. At least 80 percent of such cases are due to skin irritation; less than 20 percent are the result of contact allergy.[1]

Irritant contact dermatitis is caused by a direct, local, toxic effect and does not involve immunologic mechanisms. When effects are observed within minutes of skin contact, following a brief skin exposure with a substance such as concentrated sulfuric acid, the injury is termed a chemical burn. Most industrial irritants are relatively weak, and require frequent or prolonged skin contact before visible changes are induced. As discussed above, factors that increase the qualitative degree of exposure or enhance percutaneous penetration will increase the potential for a given substance to provoke irritant contact dermatitis. Depending on the conditions under which skin exposure occurs, almost any substance encountered in the workplace may become an irritant.[20] The most commonly encountered irritants include solvents, acids and alkalis, and soaps and detergents. Workers are usually exposed to multiple potential skin irritants, and irritation may be a cumulative result of multiple exposures. Once skin inflammation has occurred, the skin barrier is compromised and the skin becomes even more susceptible to irritation. Abrasive soaps and waterless hand cleaners (usually containing 10 percent to 20 percent petroleum distillate in a lanolin or cream formulation), are additional potential irritants.

Once irritation has occurred, these skin cleaners may do more harm than good.[24]

Allergic contact dermatitis results from immunologic mechanisms involving cellular delayed hypersensitivity. Most allergens encountered in industry are relatively weak, and require optimum circumstances of exposure in susceptible persons before sensitization occurs. This usually takes time, and months (or years) may pass before a worker actually has an allergic reaction. Thus, allergy often develops to longstanding contacts in a worker's environment, rather than recently introduced substances. Common occupational sensitizers include metal salts (nickel, chromium, cobalt, gold); rubber accelerators and antioxidants; epoxy, acrylic and phenolic resins; biocidal agents used to preserve aqueous solutions or emulsions (for example, cutting fluids), and organic dyes (such as paraphenylenediamine).[25] Domestic sensitizers (fragrances, cosmetic preservatives and topical medications) may find their way into the workplace in the form of soaps, hand creams and first aid cabinet preparations. Once allergic sensitization has occurred, only minimal quantitative exposure may be necessary to sustain dermatitis.

Photosensitivity

The activation of a chemical substance on the skin surface by ultraviolet or visible light causes a photosensitive reaction. These clinically appear as an acute sunburn or eczema on sun-exposed surfaces of the body. Phototoxic reactions are analogous to irritant contact dermatitis, and do not involve immunologic mechanisms. Polycyclic aromatic hydrocarbons, such as creosote[26] and tar, cause burning and stinging cutaneous reactions following sun exposure. Severe, bullous, phototoxic reactions occur in celery harvesters, caused by a furocoumarin liberated from celery parasitized by a common agricultural fungus ("pink rot").[27] The newer polyfunctional, ultraviolet-light-cured acrylic resins may produce phototoxic as well as photoallergic reactions.[28] Persistent photosensitivity has occurred in workers developing photoallergy to vaporized epoxy resin.[29]

Acne and Chloracne

Lubricating oils and greases may cause comedonal plugging or a pustular folliculitis (oil acne).[30] This usually occurs on the hands and dorsal forearms, but may be particularly severe on covered areas of the body if clothing becomes saturated. Insoluble cutting oils and various other petroleum oils and greases encountered in machining operations and automotive repair work are common causes.

Chloracne is distinct from oil acne. The former may be caused by exposure to various chlorinated or halogenated naphthalenes, diphenyls, azoxybenzenes, dibenzofurans and dioxin.[31] Chloracne is characterized by a predominance of closed comedonal lesions (although some inflammatory lesions may be present), and is usually associated with the presence of noninflammatory, pathognomonic, straw-colored cysts. Involvement of the cheeks, temples, ears and postauricular areas of the face is most common, but covered areas may be involved in severe cases. Occurrences have usually been associated with massive exposures, such as industrial accidents or contamination during manufacture. The disease has its usual onset within months following initial exposure. Although acquired lesions may persist and are relatively resistant to treatment, new lesions usually cease to form within 6 to 12 months after exposure has stopped. Chloracne is a sensitive index of exposure, and is present in up to 95 percent of cases where systemic symptoms (liver disease, neurologic changes) are present.[32]

Pigmentary Disturbances

Slate gray or melanotic discoloration of the skin may result from systemic absorption of various heavy metals, such as silver, mercury or arsenic. Direct chemical staining of the skin may occur and serves as a useful marker of exposure when systemic symptoms are associated. Nitrite compounds, such as nitric acid, trinitrotoluene and dinitrophenol produce yellow stains, presumed to arise from nitration of aromatic nuclei of certain amino acids incorporated into cutaneous protein.[33] Explosive or abrasive forces may cause adventitial tattooing by embedding pigmented foreign material in the skin[34]; "coal miner's tattoo," a blue-gray discoloration from inoculation of coal dust, is an example.[35]

Nonspecific postinflammatory hyperpigmentation may follow any episode of dermatitis.[36] Photosensitizers such as tar, pitch or furocoumarins may increase pigmentation without obvious antecedent inflammation.[37] Reversibility occurs much more slowly in darkly complexioned persons than in whites. Hypopigmentation or depigmentation may also result from nonspecific postinflammatory changes, following dermatitis or thermal burns.[36]

Depigmentation resembling idiopathic vitiligo can be caused by occupational exposure to a variety of phenolic or catecholic derivatives.[38,39] These chemicals bear a structural resemblance to tyrosine and may selectively inhibit melanin synthesis; at higher doses they are cytotoxic to melanocytes. A partial list includes p-tertiary butylphenol, p-tertiary butylcatechol, p-tertiary amylphenol, hydroquinone and monobenzyl ether of hydroquinone. They serve as antioxidants or germicidal disinfectants and may be encountered with exposure to rubber, photographic developing solutions, lubricating oils, plastics or adhesive manufacture, and hospital, institutional or industrial disinfectant cleaning solutions. Depigmentation may or may not be preceded by inflammatory changes.

Urticaria

By comparison to occupational asthma, occupational urticaria is rare. Generalized urticaria, when caused by occupational exposure, usually results from inhalation of allergenic material accompanied by other symptoms of inhalant allergy. A partial list of exposures includes castor bean pomace, platinum salts, spices, penicillin, formaldehyde, aliphatic polyamines, ammonia, sulfur dioxide and lindane.[14]

Localized urticarial reactions (both immunologic and nonimmunologic) may result from exposure to a variety of substances following percutaneous absorption. This reaction has been dubbed "contact urticaria."[15] Rarely, urticaria may become generalized. The list of substances that can provoke this reaction is constantly expanding. Some examples include flavoring and fragrances (cinnamic aldehyde), spices, antibiotics (penicillin, bacitracin), plant materials (nettles) and various proteinaceous food substances (meat, vegetables).[15] Food handlers are particularly susceptible, possibly because the skin barrier is easily damaged by water and irritating juices, facilitating percutaneous penetration.

Neoplastic Disease

The role of aromatic hydrocarbons such as coal, tar, mineral oil, lubricating oil, pitch and creosote in the induction of cutaneous papillomas, keratoses and malignant conditions has been recognized for two centuries. Percival Pott first suggested in 1775 that soot caused scrotal cancer in chimney sweeps. In 1792 tar was implicated in lip cancer. Inorganic arsenic was subsequently recognized as a carcinogen of the skin and other organs. All require a cocarcinogen for induction of neoplastic disease, of which ultraviolet light in the case of coal tar derivatives and trauma in the case of chimney sweeps are the most widely accepted.[40]

Ionizing and ultraviolet radiation may induce a radiodermatitis and neoplastic transformation, following a latency of several years.[41] Despite the large body of supporting evidence, workers' compensation boards have been reluctant to accept claims. The latent period between exposure and onset of disease, coupled with shifts in employment, complicate the issue of establishing liability to a single employer. The economic impact of accepting all skin cancer in outdoor workers as occupational disease would probably be disastrous to most insurance carriers.

The exact role of cutaneous trauma in the induction of neoplastic disease remains controversial. Skin cancer arising directly within an area of trauma has been documented on numerous occasions.[42,43] From a scientific viewpoint, evidence suggests that trauma is a cocarcinogen rather than a true carcinogen.[40]

A specific cutaneous lymphoma, mycosis fungoides, has been linked epidemiologically to work in manufacturing industries.[44] Evidence suggests that, in some cases, mycosis fungoides may evolve from chronic bouts of allergic contact dermatitis,[45] with subsequent malignant T-cell transformation.

Vascular Disease

Raynaud's phenomenon, a vasospastic disorder, may be induced in workers who use hand-held vibratory tools such as chain saws; this has been termed "traumatic vasospastic disease" or "white finger disease."[46] The responsible frequency is in the 125 Hz range, which corresponds roughly to the most sensitive frequency to which pacinian corpuscles respond.[47] Reflex linkage of chronic stimulation of the pacinian corpuscles to the sympathetic innervation of the cutaneous vasculature may be involved. The disease correlates with the duration of use of vibratory equipment, and usually does not appear until at least two years after a worker has begun to use vibratory tools.[48]

Acro-osteolysis, characterized by resorption of the distal phalanges of the hands in association with Raynaud's phenomenon, occurs in workers exposed to free vinyl chloride monomer, usually by cleaning reactors where polymerization to polyvinyl chloride is carried out.[49] Occupational scleroderma-like disorders, associated with Raynaud's phenomenon, have occurred from exposure to silica dust, expoxy resin hardener and perchloroethylene.[50] Similar disorders are alleged to be caused by a variety of pesticide exposures but the evidence is unclear.

Granulomatous Reactions

Foreign body granulomas usually present no difficulty in diagnosis. Beryllium and zirconium, when inoculated into the skin, have produced allergic granulomatous reactions; chemically induced granulomas from talc and silica have the appearance of a granuloma caused by a foreign body and do not induce hypersensitivity.[51]

Disorders of the Hair and Nails

Occupational exposure to thallium-containing rodenticides, boric acid, arsenic and chloroprene may cause a diffuse toxic alopecia[52]; hair regrowth usually starts within a few months following cessation of exposure. Ionizing radiation may similarly induce an acute toxic alopecia.

Like the skin, the hair and nails are subject to trauma and may become discolored from exposure to various dyes and other materials. Absorption of pesticides into the nail folds may result in nail plate deformation and altered growth.[53]

Disorders Caused by Infectious Agents

Poor skin hygiene may nonspecifically predispose a worker to a variety of skin infections. Abrasions and lacerations may become infected, leading to cellulitis, deep fungal infections or pyoderma. Infections associated with specific occupations include anthrax in sheep handlers and wool workers, erysipeloid in meat and fish handlers and sporotrichosis in gardeners and horticulturists. Herpetic whitlow, caused by inoculation of herpes virus into the skin of the finger, may affect nurses and dental assistants who contact oral secretions. However, a recent study, which showed that 11 of 13 cases of herpetic whitlow were caused by type II virus, suggests a venereal epidemiology for most of these infections.[54]

Disorders Caused by Physical and Mechanical Agents

Excessive heat, cold, wind, vibration and radiation (ultraviolet and ionizing) may cause a variety of direct or indirect cutaneous changes. Examples include thermal burns, frostbite, cutaneous chapping, sunburn, and acute and chronic radiodermatitis. Sweat retention under working conditions of excessive heat and humidity may result in inflammation of the sweat glands, miliaria ("prickly heat"). Vasospastic disease from vibratory tools has been discussed above.

Warm, dry air may cause generalized pruritus due to the drying effect of low humidity in the skin.[55] Large-scale outbreaks of nonspecific pruritus are not uncommon in working environments where low humidity is an important quality control measure, as in semiconductor manufacturing and electronic assembly operations.

Friction and pressure may lead to blister or callus formation on the hands of laborers. Fiberglass produces pruritus from mechanical irritation, particularly in flexural areas where the fibers may be trapped beneath clothing and rubbed against the skin. Visible cutaneous changes are usually the result of rubbing and scratching, rather than a direct effect of the fiberglass. Pruritus is directly related to fiber diameter and inversely proportional to fiber length.[56] Beta fibers are thinnest and are much less irritating.[57]

Establishing a Diagnosis

The examining physician must determine if the disease under evaluation directly resulted from occupational exposure, was unrelated or was substantially aggravated (although not primarily caused) by such exposure. The decision is based not only on medical information with which the physician is familiar but also on technical information concerning industrial processes and job performance. The physician must identify the cause as precisely as possible so that appropriate steps to avoid such exposure may be instituted and time lost from work minimized. The patient is often unable to furnish accurately all the necessary information, and telephone calls to appropriate supervisors and safety personnel, or a visit to the workplace, may be necessary. Once a list of exposures has been obtained, further identification from raw material manufacturers and technical or toxicologic information may be needed. This information may be unintentionally misleading, due to suboptimal toxicology testing procedures or inaccurate laboratory analyses. Calnan cites several such examples.[58]

During the physical examination, the physician must distinguish primarily acquired occupational skin disease from endogenous conditions (for example, atopic dermatitis or psoriasis) that may be aggravated by work, or other unrelated disorders (such as scabies). A complete examination of the entire skin surface is usually necessary. Considerable dermatologic skills and experience may be required.

The patch test is used for precise allergen identification when allergic contact dermatitis is suspected. Details of the methodology may be found in any textbook on the subject.[25] In principle, suspected allergens are occluded and are held in contact with the skin by adhesive tape for 48 hours. Localized, eczematous reactions occur within two to four days at the site of patch test application if the person is allergic to the tested substance. Because 30 percent to 40 percent of all reactions do not become positive until 72 to 96 hours after application, delayed readings are necessary.[59] Any deviations from accepted guidelines may give false positive or negative reactions.

A routine patch test screening series, available through the American Academy of Dermatology and other sources, tests only the most common, not all, allergens. Although it is extremely useful in the detection of domestic contact allergy, many allergens encountered in the workplace (gold, acrylic resins, phenol formaldehyde resins and numerous industrial biocidal agents) are not included. Unless specifically tested, these allergens may be overlooked.

Prevention

Methods to prevent occupational skin disease include protection or isolation of the worker from his environment, replacement of allergenic or irritant substances with technically suitable alternatives and removal of the worker from the work environment.[60] Removal of the worker should be considered the least desirable alternative, and should not be undertaken lightly.

Protection of the Worker

Protective clothing and barrier creams, good skin hygiene, good housekeeping and process containment through appropriate engineering controls are all means of protecting the worker. Protective clothing (gloves, boots, sleeves, aprons, coveralls and face gear) is available in a variety of fabrics; care must be taken that they work properly so that potentially noxious substances are not trapped and occluded against the skin. From a scientific viewpoint, the effect of barrier creams appears negligible.[61] However, the lubricating effects of barrier creams, when combined with a good skin hygiene program, may be beneficial.[24]

Irritant and Allergen Replacement

Contact dermatitis may sometimes be successfully managed simply by replacing the offending irritant or allergen with a suitable alternative equal to the task of the removed substance.[62,63] Substitution that is successful in eliminating dermatitis depends on precise identification of the actual irritant or allergen. Contact allergy triggered by epoxy resin may be avoided by substituting another adhesive, such as acrylic resin, when great bonding strength is unnecessary.

Future Needs

Research

Because skin disease represents 40 percent to 50 percent of all occupational illnesses, an increased emphasis on dermatologic research has been urged by the National Institute for Occupational Safety and Health[64] and others.[65] Immunological research, with emphasis on suppressor mechanisms and hyposensitization in allergic contact dermatitis, must be pursued. Mechanisms involved in causation of chloracne require further elucidation. Epidemiologic data, particularly on long-term effects of cutaneous exposures to potential carcinogens and other agents, must be developed. The sensitivity of existing toxicologic screening procedures for irritant, allergic, chloracneigenic and neoplastic potential need to be increased, and new models developed.

Education

In view of the substantial proportion of occupational illness caused by skin disease, increased participation of dermatology as a field of study in occupational medicine curricula at the undergraduate and postgraduate levels is mandatory. Present undergraduate training in most medical centers in the United States involves relatively little training in general dermatology and virtually no training in occupational medicine. Dermatology residency programs similarly lack sufficient training in diagnosis and management of occupational skin diseases.

Cooperation of Industry

Accurate technical information and assistance is essential to the management and prevention of occupational skin disease. Industry has regarded the activities of physicians, aside from those directly employed, with great suspicion. Physicians have met resistance in the pursuit of technical information or when they have requested access to workplaces. A closer working relationship among physicians, management, and industrial safety and health personnel is essential, if future needs and goals are to be met. Complete labeling of ingredients—already a fact of life in the cosmetics industry—and material safety data sheets with accurate dermatotoxicologic information are important steps in this direction.

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